GNR That Cause Diarrhea

Introduction

The organisms in this lesson are all **Gram-negative** bacteria that cause **diarrhea**. The organization of the lesson separates those that cause dysentery, a bloody diarrhea, from those that cause a watery diarrhea. In general, to get red blood cells in the stool, an organism will have to invade into the mucosa. Invasion into the mucosa by a foreign organism tends to get noticed by the immune system. Therefore, **bloody diarrhea** is a marker of **invasion**, and is commonly seen with **fecal leukocytes** (that means RBCs and WBCs in the stool). On the other hand, organisms that cause watery diarrhea tend to do that with **toxins**, meaning that the absence of blood and the absence of white blood cells in the stool is indicative of noninvasive bacteria.

The **enterotoxic** bacteria use toxins that affect the cells of the intestines, called enterocytes (entero-toxic). The **invasive** bacteria use various virulence factors to invade, and can therefore also get into the bloodstream and cause severe disease. There are, of course, exceptions, but siloing these organisms by the clinical picture they cause helps to keep them straight. We go through laboratory diagnostics, then bloody diarrhea, then watery diarrhea.

Laboratory Diagnostics

Separate the laboratory diagnostic similarities from the clinical. Memorize Table 9.1. It is an inclusive table, an organism listed only if the organism is positive for the feature. That means the rest are negative. The danger is that you follow the diagnostic algorithm and believe that because two organisms share a feature of laboratory diagnosis, they will also share features of clinical presentation of disease. Take *Vibrio cholerae* and *Campylobacter jejuni*. They are both oxidase positive, comma-shaped, Gram-negative rods, do not ferment lactose, and do not produce H_2S. *Vibrio* causes cholera, an enterotoxin-mediated watery diarrhea, while *Campylobacter* causes a bloody diarrhea and invasion of the colon without the production of enterotoxin.

Don't learn all the microbiology for all bacteria; learn the specific microbiology that matters to any one given bacterium. We call out the features here very quickly, reserving the discussion of the features in the illness scripts for the bacteria, later in the lesson.

All of these organisms are Gram-negative bacteria. Gram-negative bacteria can be differentiated by their ability to ferment lactose. They were discussed in the previous lesson. One of them was *E. coli*. *E. coli* makes an appearance in this lesson as well, which means that all organisms other than *E. coli* in this lesson are not lactose fermenters. Gram-negative bacteria can also be distinguished by whether they produce oxidase (like *Pseudomonas* in the last lesson). This is not a meaningful distinction in this lesson, as you will not use oxidase to differentiate any two organisms from one another, but you should know that the comma-shaped rods, *Vibrio* and *Campylobacter*, do produce oxidase; the rest do not. Certain organisms need to be cooked at specific temperatures in order to grow; the preferred incubation temperature that optimizes colony growth helps set them apart. *Y. enterocolitica* likes it cold; *Campylobacter* needs it hot. Finally, TSI agar turns black in the presence of colonies that produce H_2S. *Salmonella* produces H_2S; the rest do not.

	LACTOSE FERMENTERS	OXIDASE POSITIVE	PRODUCE H_2S TURN TSI BLACK	TEMPERATURE SENSITIVE
This Lesson	*E. coli*	*Vibrio* *Campylobacter*	*Salmonella*	*Campylobacter* grows better at 42°F *Y. Enterocolitica* grows better at 25°C
Don't forget	*Klebsiella*	*Pseudomonas*	*Proteus*	

Table 9.1: Laboratory Diagnosis of Diarrhea Bugs

Bloody Diarrhea

The following organisms—*Shigella*, bloody *E. coli*, *Campylobacter*, and *Y. enterocolitica*—cause bloody diarrhea. The common themes will be infection of the **colon** and **invasion** of the mucosa (not necessarily invasion through the mucosa, but at least into the mucosa between epithelial cells), which together cause **bloody diarrhea**. Some organisms are deemed important enough to merit separate paragraphs (e.g., on their Structure, Virulence, Epidemiology, etc.). These are organisms whose details should be learned. Other organisms are treated in a single catch-all paragraph. The details are less necessary for those, or their mechanisms are discussed elsewhere.

Shigella

Shigellosis, the disease caused by *Shigella*, is a common self-limiting bloody diarrheal disease that happens to kids. *Shigella* produces Shiga-toxin and can lead to HUS. HUS stands for hemolytic uremic syndrome, characterized by microangiopathic hemolytic anemia, renal failure (uremia), and thrombocytopenia, a syndrome discussed in greater detail in Heme/Onc: Clotting #4: *Platelet Bleeding*. Shigella-Pediatric-Shiga-HUS is generally benign and needs only supportive treatment, unlike the EHEC-Adult-Shiga-HUS which is more like thrombotic thrombocytopenic purpura (TTP).

Microbiology. *Shigella* is non-lactose-fermenting, so is colorless on MacConkey agar. It is oxidase negative, and does not produce H_2S, so is not black in color on TSI agar. It is immotile.

Virulence. *Shigella* requires only 100–1,000 organisms to cause an infection, which is an **extremely low infectious dose**, meaning person-to-person transmission is possible, which is why fecal-oral transmission is possible. It is also **acid-stable**, resistant to gastric acid.

Epidemiology. Humans are the only reservoir for *Shigella*. It is transmitted fecal-oral, person to person. The organisms that frequently touch their butt, their mouth, and other organisms' mouths are children. Epidemics occur in day care centers. Shigellosis is primarily a pediatric disease. Because of its low infectious dose, places with poor sanitation are at highest risk. The CDC lists men who have sex with men as being at high risk, though any activity—sexual or not—involving anyone's anus can increase the risk of transmission.

Disease. *Shigella* causes dysentery and the Shiga toxin can cause HUS (Bacteria #3: *Toxins*). After ingestion and passage through the stomach, *Shigella* invades through the **M cells** of Peyer's patches. These cells are designed to sample the lumen for antigens. Waiting on the other side of the M cell in the lamina propria are macrophages. Macrophages phagocytose *Shigella*. But *Shigella* **lyses the phagosome** and escapes into the cytoplasm, where it replicates. The macrophage dies; the *Shigella* is released, and infects the epithelial cells. It is also able to **move between epithelial cells** (cell-to-cell transmission). The bacteria re-enter the lumen, re-enter the M cells, and re-enter macrophages (well, new ones). Each time a macrophage engulfs a bacterium, it sends out inflammatory cytokines,

recruiting neutrophils to the site of infection. The reinfection causes the death of the epithelial cells, leading to ulceration of the mucosa. The presence of many neutrophils is called pus. The cardinal features of shigellosis are lower abdominal cramps and straining to defecate, then **abundant pus and blood in the stool**. It is usually self-resolving, and *Shigella* never gets to the bloodstream; no antibiotics are needed. The **Shiga toxin** does get absorbed by the gut, and circulates throughout the bloodstream. The Shiga toxin can cause HUS. The diagnosis of shigellosis is made by finding the toxin or DNA for the toxin, and not by culturing or serotyping the organism.

Bloody *E. coli*

Two *E. coli* strains cause **bloody diarrhea**—EIEC and EHEC. Enteroinvasive *E. coli* (EIEC) is one of those strains, but is simply too rare in developed countries to spend much time on. You should merely be aware that EIEC exists. Because I is next to H in the alphabet, it also helps trigger a memory cue for the other bloody *E. coli*, EnteroHemmorhagic *E. coli* (EHEC). EHEC causes bloody diarrhea and can result in hemolytic uremic syndrome, like *Shigella*, because both *Shigella* and EHEC produce Shiga toxin. Shiga Toxin *E. coli* (STEC) is the new name for EHEC (technically, EHEC is a subset of STEC, but for you they are the same thing). EHEC invades **colonic enterocytes**, producing **hemorrhagic colitis**. The bloody diarrhea is caused by the death of enterocytes.

EHEC/STEC produces Shiga toxin. The famous serotype ***E. coli* O157:H7** caused a rash of diarrheal syndromes at a fast-food chain across the United States, a product of undercooking frozen beef hamburgers. While O157:H7 is the most common serotype to cause human disease, it accounts for only half of the human disease caused by STEC serotypes. Therefore **diagnosis is based on the presence of Shiga toxin**, not on the serotyping of the *E. coli*. PCR of the stx1 or stx2 gene (Shiga toxin) confirms the diagnosis.

EHEC/STEC does **not ferment sorbitol**, separating it from all other *E. coli* on culture (this factoid is easily tested, though we just stated diagnosis is based on the toxin, not the organism). **Do not give antibiotics**. Symptoms start around day 3 and end in days 6–10. If you were reading correctly, you may have noticed that bloody diarrhea from *Shigella* and EHEC are both diagnosed by finding Shiga toxin, do not need antibiotics, and may cause HUS.

Yersinia enterocolitica

Yersinia enterocolitica is a *Yersinia* species. It shares the genus with the organism that causes bubonic plague, *Yersinia pestis,* discussed in Bacteria #10: *GNR Transmitted by Animals*. We want you associating "*Yersinia*" with "Plague." *Yersinia entero-col-itica* causes infections of the intestines (entero) and colon (coli) leading to inflammation ("-itica"). Therefore, to emphasize the disease it causes and to separate it from plague, we will refer to this bug only as only as *Y. enterocolitica*.

Y. enterocolitica is a Gram-negative rod. *Y. enterocolitica* is spread in **contaminated food** because it requires a **large inoculum** to cause disease. The source is usually domesticated animal feces (classically puppies), contaminated **pork** (never eat raw pork), or unpasteurized milk. Its only notable microbiological property is that it **likes cold temperatures**. It **grows at 4°C**, so refrigeration is inadequate to prevent contaminated food from growing colonies. Likewise, and unlike most bacteria, when trying to culture it in the lab, *Y. enterocolitica* grows better at room temperature than at body temperature, so should not be incubated when attempting to culture it. Because it has no unique features, it does not ferment lactose, is oxidase negative, and does not produce H_2S or black pigment on TSI agar.

Y. enterocolitica causes a bloody diarrhea. It is an invasive organism that invades the terminal ileum and the colon. That invasion is what provokes the bloody diarrhea. But inflammation provoked by this infection can cause the abdominal lymph nodes to swell, leading to a right lower quadrant abdominal pain that presents similarly to appendicitis, called **pseudoappendicitis**. This is actually a presentation of terminal ileitis (inflammation of the ileum) and abdominal adenitis (inflammation of the lymph nodes). Since *Y. enterocolitica* infections tend to occur in children (who play with their pet and therefore their pet's poop) and self-resolve, making sure that it is not appendicitis is pretty important. Ultrasound is chosen, given that children are the usual victims.

Campylobacter

Campylobacter is the most common cause of bacterial gastroenteritis in both developing and developed countries, with *Campylobacter jejuni* responsible for most cases. We will explore only *jejuni* (which gets its name from the jejunum, which it infects), and so will say only *Campylobacter* from here out.

Physiology/structure. *Campylobacter* is a **curved**, comma-shaped, Gram-negative rod; it is oxidase positive, motile, and microaerophilic (growing best in reduced oxygen). *Campylobacter* **grows best at 42°C** and does **not grow at 25°C**. That means the incubation must occur in warmed isolation and not at room temperature (22°C). It is the only bacterium we will teach you that does this. *Campylobacter* has a polysaccharide capsule. Although it does secrete toxins, the role of those toxins in human disease is uncertain.

Epidemiology. *Campylobacter* is ubiquitous, causing bacterial diarrhea around the world. It is spread **in contaminated food** because it requires a **large inoculum** to cause disease. Contaminated foods are **poultry** or **unpasteurized milk**.

Disease. *Campylobacter* causes an **invasive enteritis**, resulting in ulceration of the mucosa of the jejunum, ileum, and colon. It is not resistant to the gastric pH, and any condition which raises the pH (antacids, PPIs) decreases the required infectious dose. The patient suffers from a **bloody diarrhea**, abdominal pain, and systemic inflammatory symptoms—**fever**. There may be up to ten bowel movements per day, though in immunocompetent hosts, even neonates, *Campylobacter* rarely goes bacteremic. Instead, extraintestinal manifestations include both **Guillain-Barré syndrome** (an ascending paralysis secondary to anti-myelin antibodies) and **reactive arthritis** (joint pains related to infection without the bacteria actually infecting the joints). It is normally self-limiting and does not require antibiotics. If antibiotics are chosen, **azithromycin** is the drug of choice. Fluoroquinolone and penicillin resistance has increased in *Campylobacter*, limiting the empiric use.

BACTERIUM	NOTES
Shigella	Primarily a disease of childhood, **epidemics in daycares**, fecal-oral only route of spread **Extremely low infectious dose** (100). It is also **acid-stable**, resistant to gastric acid. **Invades** through **M cells** of Peyer's patches, **lyses phagosome** of macrophage Abundant blood and pus in stool after abdominal cramps, straining to defecate **Shiga toxin** causes hemolytic uremic syndrome Diagnosis is made by the presence of **Shiga toxin** or PCR for Shiga toxin genes Self-limiting, may cause HUS
Bloody *E. coli*	Primarily a disease of childhood **Extremely low infectious dose** (100–1,000) EnteroHemorrhagic *E. coli* (EHEC) and EnteroInvasive *E. coli* (EIEC) infect the **colonic enterocytes** EHEC is also Shiga-Toxin *E. coli* (STEC); does **not ferment sorbitol** **Shiga toxin** causes the same syndrome as *Shigella*; EHEC found in **undercooked beef** Diagnosis is made by the presence of **Shiga toxin** or PCR for Shiga toxin genes Self-limiting, may cause HUS
Yersinia enterocolitica	**Contaminated food** = pork, **high inoculum** (also puppy feces) Can be transmitted in raw pork or unpasteurized milk The terminal ileum and colon (entero-coli) are affected **Pseudoappendicitis** (RLQ abdominal pain, not the appendix) from inflamed lymph nodes of mucosa Self-limiting, bloody diarrhea
Campylobacter	**Most common** cause of bacterial diarrhea worldwide **Contaminated food** = **poultry**, unpasteurized milk, or children who eat their pets' poop Affects neonates and children, fecal-oral Grows at 42°C, **does not grow at 25°C**, microaerophilic, comma-shaped Gram-negative rod Invasive, self-limiting, bloody diarrhea (Azithromycin if severe) **Guillain-Barré** and **reactive arthritis** are sequela

Table 9.2: Bacteria That Cause Bloody Diarrhea

Watery Diarrhea

The following organisms—*Salmonella*, watery *E. coli*, and *Vibrio* species—cause watery diarrhea.

Salmonella Other-Than-Typhi

Salmonella isn't just *Salmonella*; there are hundreds of species and serotypes. But when it comes down to what you need to know there are only two salmonellas—*Salmonella* Typhi that causes typhoid fever, and every other species that doesn't. This first section is about the *Salmonella* species that do not cause typhoid fever. They cause a watery diarrhea or osteomyelitis in sickle cell patients (again, gross oversimplification, but the fact is, that's all you need to know).

Physiology/structure. *Salmonella* is a Gram-negative rod. *Salmonella* is oxidase negative. It **produces H_2S** and so turns TSI agar black. It is **motile** (salmon swim, *Salmonella* swims too).

Virulence. *Salmonella* Not-Typhi requires a **high infectious dose** to cause infection and therefore is spread in **contaminated food.** *Salmonella* isn't very good at infecting its host, but if it is able to infect, it

causes some serious problems. *Salmonella* can cause an **infection of the GI tract**, but it can also **spread hematogenously**. *Salmonella* has the most success when contaminated food (large dose of organisms) is consumed by a person with low stomach acid (so a higher-than-normal pH).

Epidemiology. *Salmonella* is why you do not prepare your vegetables on the same surface where you dice your chicken. The most common sources of human infections are **poultry**, **eggs**, and dairy products. Raw chicken on a wooden cutting board allows the *Salmonella* to get into the wood. After you put your chicken in the oven, cooking the chicken and killing the *Salmonella*, the *Salmonella* left behind on the cutting board is still alive and well. Add to it some nice lettuce (which you are obviously not going to cook), and the *Salmonella* hops on the lettuce. You serve the lettuce with the cooked chicken, and are infected by the *Salmonella*-contaminated lettuce. Animal pets can serve as reservoirs, and **pets with eggs** can infect their owners (turtles, snakes). The real-life concern is raw poultry and eggs; the board examinations have snakes and turtles.

Raw poultry and bloody diarrhea is *Campylobacter*; raw poultry and watery diarrhea is *Salmonella* Not Typhi.

Disease. The diseases *Salmonella* Not Typhi causes are enterocolitis and disseminated disease. After ingestion and passage through the stomach, salmonellae attach to the mucosa of the **small intestine** and invade into the **M cells** of Peyer's patches. Yes, just like *Shigella*. Just like how many bacteria invade the mucosa. What comes next is very unlike *Shigella*. *Salmonella* Not Typhi **prevents fusion** of the lysosome, replicates in the phagosome, but does not go cell to cell, does not have repeated cycles of infection, nor does it cause a bloody diarrhea.

Enterocolitis. If the host immune response is good, inflammation contains the bacteria to the mucosa, mediates release of prostaglandins, and stimulates cAMP to actively secrete fluid. This causes **nausea, vomiting,** and a **watery, nonbloody diarrhea**. Even though it is invasive to the Peyer's patches, most are not bloody. This is taken from *Medical Microbiology* (a textbook) though it seems to be in contrast to many review resources that report *Salmonella* as a bloody diarrhea. There is no need for antibiotics, and in fact, antibiotics **prolong the duration**.

Disseminated disease. If the host immune response is poor, **bacteremia** may result. Specifically, this is the *Salmonella* that causes **osteomyelitis** in **sickle cell patients**. While more severe forms of disseminated disease can occur (bacteremia, meningitis), we want you learning, "bone infection, sickle cell disease, *Salmonella* Not Typhi." Treatment is with **ceftriaxone**.

Salmonella Not Typhi does not cause typhoid fever. *Salmonella* Typhi (discussed next) causes typhoid fever. Typhoid means typhus-like. Typhus (sometimes typhus fever) is caused by *Rickettsia* species transmitted by a tick (discussed in Bacteria #12: *GNR: Intracellular Obligate Parasites*), which presents with a fever and a rash. Typhus and typhoid have nothing to do with diarrhea, and everything to do with fever and a rash.

Salmonella Typhi

Salmonella enterica serotype Typhi ("Salmonella Typhi") causes typhoid fever. Typhoid Mary (Mary Mallon) was the quintessential case of *Salmonella*, the first person to be identified as an asymptomatic carrier, and thought to have infected 50 people with it. Her case illustrates a course of the disease that rarely occurs—the silent carrier shedding *Salmonella* Typhi into the intestines from the gallbladder.

Salmonella Typhi shares all the same features of *Salmonella* Not Typhi, but adds a few. First, the **infectious dose is very small**, so person-to-person transmission is possible. Second, there are **few to no diarrheal symptoms**. Instead, there is **enteric fever** (the noneponym for typhoid fever).

Salmonella Typhi bacteria are brought to the spleen, liver, and bone marrow by their macrophage "captors." *Salmonella* Typhi replicate. Two weeks after ingestion they go bacteremic. The patient experiences gradually increasing fever, with nonspecific complaints of headache, myalgias, malaise, and anorexia. The **rose spots on the trunk** represent dissemination. These rose-colored spots and fever resemble endemic typhus. The bacteria get into and colonize the gallbladder, where they live, replicate, and are shed into the lumen, passed on to the next victim in stool. Treatment of *Salmonella* Typhi is usually with fluoroquinolones such as ciprofloxacin, though colonized patients are often very difficult to clear. Instead, good sanitation practices (washing hands) avoids infection.

Watery Diarrhea *E. coli*

There are three *E. coli* strains that cause watery diarrhea, but only one that is seen in the United States. All three cause **infant diarrhea in developing countries** and all three present as a **self-limiting, non-bloody, watery diarrhea**. All three have a site of action in the **small intestine** (compared with the bloody diarrhea *E. coli*, which infects the colon). Enteroaggregative *E. coli* (EAEC) and Enteropathogenic *E. coli* (EPEC) you will not hear mentioned again. EnteroToxic *E. coli* (ETEC) is what we will focus on. It is known as Traveler's diarrhea.

ETEC is one of the most common causes of bacterial diarrheal disease in developing countries. There are an estimated 850 million cases annually, and ETEC is implicated in an estimated 30% of travelers to these countries who develop diarrheal disease. The **inoculation dose is very high—**there must be a large number of bacteria to cause disease. Because of this, person-to-person spread does not occur. Instead, it is from **contaminated water—**water with feces in it. Places at highest risk have poorly developed sanitation systems. After a two-day incubation period, symptoms (watery, nonbloody diarrhea; abdominal cramps) last for about 3–5 days. No antibiotics are required.

ETEC grabs on to the epithelium of the small bowel using proteins called colonization factors. It does not invade the mucosa. It the elaborates two toxins—heat-labile and heat-stable exotoxins. These were discussed in detail in Bacteria #3: *Toxins*. The result is increased secretion by enterocytes, which tips the balance in favor of secretion over absorption. The toxin's mechanism is the same as the cholera toxin, though the symptoms, the volume of water secreted, tends to be much higher in cholera.

The parallels between ETEC and *Vibrio cholerae* are the mechanism of toxin, symptoms of watery diarrhea, and the requirement of a large inoculum to cause disease.

Vibrio

Cholera is caused by *Vibrio cholerae*. The majority of what you need to know about cholera was covered in the toxins lesson. *Vibrio cholerae* secretes an enterotoxin, an AB toxin that turns on G_S via ADP-ribosylation, leading to **rice-water stools**—lots of water, not a lot of stool. We discuss this secretory diarrhea and its mechanisms in substantial detail in GI: Digestion and Absorption: Start to Finish #13: *Functional Intestinal Diseases*. Here, we discuss a little bit about *Vibrio cholerae*'s microbiology and two of its cousins.

Vibrio cholerae is a curved, **comma-shaped** bacterium (as is *Campylobacter*). It is **motile** with a **single flagellum**. It, like campylobacter, is **oxidase positive**. It is transmitted fecal-oral via contaminated water or seafood. *Vibrio cholerae* is not a hardy bug. It is **acid-labile** and requires a **large inoculum** to cause disease. People with intact immune systems and normal gastric emptying times (people who do not already have diarrhea) can generally destroy enough *Vibrio cholerae* to prevent disease, if it is ingested. Those who have **reduced gastric acid** (so a higher than normal pH) are at a greater risk of having the disease. *Vibrio cholerae* produces mucinase, which allows it to adhere to the mucosa. Cholera toxin and cholera symptoms are an exaggerated version of ETEC toxins and symptoms.

Vibrio parahaemolyticus and ***Vibrio vulnificus*** are cousins of *Vibrio cholerae*. *V. parahaemolyticus* causes a gastroenteritis, like *Vibrio cholerae* does, OR it causes havoc to wounds, like *V. vulnificus*. *V. vulnificus* causes severe, fatal infections characterized by erythema, pain, bullae, and tissue necrosis. Those at greatest risk are those with chronic liver disease. *V. parahaemolyticus* and *V. vulnificus* are associated with shellfish. **Cirrhotics should never eat raw shellfish.**

BACTERIA	NOTES
Salmonella Typhi	**Fecal-oral** transmission, infects macrophages, prevents fusion of lysosome Carried to liver, spleen, bone marrow, where it replicates, causing typhoid fever Extremely low infectious dose Typhoid fever—**bacteremia, fevers**, and **rose spots on trunks**—but no diarrhea Bacteremia distributes bacteria to the intestines (infectious) and invades in **gallbladder** Carrier state in gallbladder, infects others, not symptomatic herself (Typhoid Mary)
Salmonella (other)	**Contaminated food** (**eggs**, **poultry**, pets that lay eggs) Infects M cells of Peyer's patches, prevents fusion of lysosome Huge infectious dose required If contained, presents with watery diarrhea; self-resolving, antibiotics prolong shedding If not, causes osteomyelitis in sickle cell; treat with ceftriaxone.
Watery *E. coli*	EnteroToxic *E. coli* (ETEC) causes Traveler's diarrhea and produces enterotoxins Large inoculum required for disease, contaminated water largest source Extremely common cause of diarrhea in developing countries and travelers to those countries **Self-limiting. No treatment is needed**—do not give antibiotics; do give hydration. Heat-labile and heat-stable toxins provoke milder cholera symptoms.
Vibrio cholerae	**Rice-water diarrhea** Large inoculum required for disease, contaminated water largest source **Destroyed by stomach acid** **Self-limiting. No treatment is needed**—do not give antibiotics, do give hydration.
Vibrio vulnificus & *parahaemolyticus*	Oysters, sea water, cirrhotics die from it Cirrhotics should avoid shellfish and never touch raw shellfish.

Table 9.3: Bacteria That Cause Watery Diarrhea

SCENARIO	ORGANISM
Bloody diarrhea and poultry	*Campylobacter*
Bloody diarrhea and unpasteurized milk	*Y. enterocolitica, Campylobacter*
Bloody diarrhea and raw pork	*Y. enterocolitica*
Bloody diarrhea and daycares	*Shigella*
Bloody diarrhea and MSM	*Shigella*
Watery diarrhea and poultry	*Salmonella*
Watery diarrhea and eggs	*Salmonella*
Rice-water diarrhea	*V. Cholerae*
Watery diarrhea while traveling	ETEC

Table 9.4: Organisms by Scenario